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<u>L7</u>	(cytokine) same (photodynamic)	47	<u>L7</u>
<u>L6</u>	(cd30L or cd30 adj ligand or cd40L or cd40 adj ligand) and (photodynamic)	7	<u>L6</u>
<u>L5</u>	(cd30L or cd30 adj ligand or cd40L or cd40 adj ligand) same (photodynamic)	1	<u>L5</u>
<u>L4</u>	L3 same (treat\$ or therap\$ or prevent\$ or administ\$)	33	<u>L4</u>
<u>L3</u>	(cd30L or cd30 adj ligand)same (tumor\$ or tumour\$ or cancer\$)	180	<u>L3</u>
<u>L2</u>	L1.clm.	7	<u>L2</u>
<u>L1</u>	(cd30 or cd30 adj ligand)same (tumor\$ or tumour\$ or cancer\$)	528	<u>L1</u>

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L4: Entry 3 of 33

File: PGPB

Sep 4, 2003

DOCUMENT-IDENTIFIER: US 20030165531 A1  
TITLE: Flt3-ligand as a vaccine adjuvant

Abstract Paragraph (1):

Flt3-ligand can be used to generate large numbers of dendritic cells from hematopoietic progenitor and stem cells. Flt3-ligand can be used to augment immune responses in vivo, and expand dendritic cells ex vivo. Such dendritic cells can then be used to present tumor, viral or other antigens to naive T cells, can be useful as vaccine adjuvants. When flt3-L is used and/or administered in combination with other reactive agents, e.g. CD40 binding proteins, 4-1BB-L or antibodies reactive with 4-1BB, CD30 ligand antagonists, RANKL, and/or interferon alpha the combination further enhances immune responses and the effectiveness of vaccine adjuvants.

Summary of Invention Paragraph (15):

[0013] The invention also provides a method of augmenting an immune response in a patient that has a cancerous or neoplastic disease wherein the method comprises the step of administering an amount of flt3-ligand sufficient to increase the patient's number of dendritic cells. Embodiments of methods for augmenting an immune response include administering flt3-ligand in combination therapies with additional active compounds, including but not limited to CD40 binding proteins, 4-1BB-L, antibodies to 4-1BB, interferon alpha, RANKL, a CD30 ligand antagonist, and combinations thereof. Such method provides a means to enhance the patient's tumor-specific immune response.

Summary of Invention Paragraph (54):

[0051] Additionally, interferon alpha, RANKL, or a CD30 ligand antagonist can be administered in combination with flt3-L to dramatically enhance immune responses. As described in Example 6, when used in a combination therapy there is a surprising synergy between flt3-L and interferon alpha for anti-tumor immune responses.

Summary of Invention Paragraph (56):

[0053] In addition to stimulating an immune response to an antigen that already exists within the patient, flt3-ligand may be administered prior to, concurrently with or subsequent to administration of an antigen to a patient for immunization purposes. Thus, as a vaccine adjuvant, flt3-ligand can generate large quantities of dendritic cells in vivo to more effectively present the antigen. The overall response is a stronger and improved immune response and more effective immunization to the antigen. Further, flt3-L may be administered as a vaccine adjuvant in combination with additional active compounds prior to, concurrently with or subsequent to administration of an antigen to a patient for immunization purposes to enhance an immune response against tumor, viral or bacterial antigens. For example, CD40 binding proteins, such as CD40-L and antibodies to CD40 which enhance the ability of dendritic cells to present antigens to T cells can be administered in combination with flt3-L to dramatically enhance an immune response. Similarly, 4-1BB-L, antibodies reactive with 4-1BB, interferon alpha, RANKL, or CD30 ligand antagonists can be administered in combination with flt3-L to enhance an immune response and provide more effective immunization to the antigen.

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L4: Entry 4 of 33

File: PGPB

Jul 31, 2003

DOCUMENT-IDENTIFIER: US 20030144187 A1

TITLE: OPG FUSION PROTEIN COMPOSITIONS AND METHODS

Summary of Invention Paragraph (6):

[0004] Therapeutic protein products have been constructed using the Fc domain to provide longer half-life or to incorporate functions such as Fc receptor binding, protein A binding, complement fixation and placental transfer which all reside in the Fc proteins of immunoglobulins. Id. For example, the Fc region of an IgG1 antibody has been fused to the N-terminal end of CD30 ligand (CD30-L), a molecule which binds CD30 receptors expressed on Hodgkin's Disease tumor cells, anaplastic lymphoma cells, T-cell leukemia cells and other malignant cell types. See, U.S. Pat. No. 5,480,981. IL-10, an anti-inflammatory and antirejection agent has been fused to murine Fc.gamma.2a in order to increase the cytokines short circulating half-life. (Zheng et al., The Journal of Immunology, 154, 5590-5600 (1995)). Studies have also evaluated the use of tumor necrosis factor receptor linked with the Fc protein of human IgG1 to treat patients with septic shock. (Fisher et al., N. Engl. J. Med., 334: 1697-1702 (1996); Van Zee et al., The Journal of Immunology, 156: 2221-2230 (1996)). Fc has also been fused with CD4 receptor to produce a therapeutic protein for treatment of AIDS. See, Capon et al., Nature, 337:525-531 (1989). In addition, the N-terminus of interleukin-2(IL-2) has also been fused to the Fc portion of IgG1 or IgG3 to overcome the short half life of IL-2 and its systemic toxicity. See, Harvill et al., Immunotechnology, 1, 95-105 (1995).

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L4: Entry 28 of 33

File: USPT

Nov 7, 2000

DOCUMENT-IDENTIFIER: US 6143869 A

TITLE: CD30 ligand oligomers and polypeptides

Brief Summary Text (64):

Preferred therapeutic agents are radionuclides and drugs. In one embodiment of the invention, the anti-tumor drug calicheamycin is attached to a soluble human CD30 ligand polypeptide.

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L4: Entry 31 of 33

File: USPT

May 19, 1998

US-PAT-NO: 5753203

DOCUMENT-IDENTIFIER: US 5753203 A

TITLE: CD30 ligand conjugates

DATE-ISSUED: May 19, 1998

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Goodwin; Raymond G.	Seattle	WA		
Smith; Craig A.	Seattle	WA		
Armitage; Richard J.	Bainbridge Island	WA		
Gruss; Hans-Juergen	Bainbridge Island	WA		

US-CL-CURRENT: 424/1.41; 424/1.69, 424/192.1, 424/193.1, 435/69.5, 435/69.7, 514/883, 530/351, 530/402

<a href="#">Full</a>	<a href="#">Title</a>	<a href="#">Citation</a>	<a href="#">Front</a>	<a href="#">Review</a>	<a href="#">Classification</a>	<a href="#">Date</a>	<a href="#">Reference</a>	<a href="#">Sequences</a>	<a href="#">Attachments</a>	<a href="#">Claims</a>	<a href="#">RIMC</a>	<a href="#">Draw Desc</a>	<a href="#">Image</a>
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☐ 32. Document ID: US 5677430 A

L4: Entry 32 of 33

File: USPT

Oct 14, 1997

US-PAT-NO: 5677430

DOCUMENT-IDENTIFIER: US 5677430 A

TITLE: Antibodies directed against CD30 ligand

DATE-ISSUED: October 14, 1997

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Goodwin; Raymond G.	Seattle	WA		
Smith; Craig A.	Seattle	WA		
Armitage; Richard J.	Bainbridge Island	WA		
Gruss; Hans-Juergen	Bainbridge Island	WA		

US-CL-CURRENT: 530/388.23; 530/387.9, 530/389.2

<a href="#">Full</a>	<a href="#">Title</a>	<a href="#">Citation</a>	<a href="#">Front</a>	<a href="#">Review</a>	<a href="#">Classification</a>	<a href="#">Date</a>	<a href="#">Reference</a>	<a href="#">Sequences</a>	<a href="#">Attachments</a>	<a href="#">Claims</a>	<a href="#">RIMC</a>	<a href="#">Draw Desc</a>	<a href="#">Image</a>
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☐ 33. Document ID: US 5480981 A

L4: Entry 33 of 33

File: USPT

Jan 2, 1996

US-PAT-NO: 5480981

DOCUMENT-IDENTIFIER: US 5480981 A

TITLE: CD30 ligand

DATE-ISSUED: January 2, 1996

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Goodwin; Raymond G.	Seattle	WA		
Smith; Craig A.	Seattle	WA		
Armitage; Richard J.	Bainbridge Island	WA		
Gruss; Hans-Juergen	Bainbridge Island	WA		

US-CL-CURRENT: [536/23.5](#); [435/252.3](#), [435/320.1](#), [435/69.5](#), [435/69.7](#), [530/351](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KIMC	Draw Desc	Image
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<u>L3</u>	(cd30L or cd30 adj ligand)same (tumor\$ or tumour\$ or cancer\$)	180	<u>L3</u>
<u>L2</u>	L1.clm.	7	<u>L2</u>
<u>L1</u>	(cd30 or cd30 adj ligand)same (tumor\$ or tumour\$ or cancer\$)	528	<u>L1</u>

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L7: Entry 9 of 47

File: PGPB

Sep 26, 2002

DOCUMENT-IDENTIFIER: US 20020136729 A1

TITLE: Combined physical and immunotherapy for cancer

Summary of Invention Paragraph (30):

[0027] The potential for combining PDT with immunotherapy was explored by Kroblik, Kros1, Dougherty and Chaplin. See Photodynamic Therapy and Biomedical Lasers, supra, at pp. 518-520. In their study, they investigated a possibility of amplification of an immune reaction to PDT and its direction towards more pervasive destruction of treated tumors. The tumor, a squamous cell carcinoma SCCVII, was grown on female C3H mice. An immunoactivating agent SPG (a high molecular weight B-glucan that stimulates macrophages and lymphoid cells to become much more responsive to stimuli from cytokines and other immune signals) was administered intramuscularly in 7 daily doses either ending one day before PDT or commencing immediately after PDT. Photofrin based PDT was employed; photofrin having been administered intravenously 24 hours before the light treatment. The SPG immunotherapy was shown to enhance the direct killing effect of the PDT. The indirect killing effect (seen as a decrease in survival of tumor cells left in situ) was, however, much more pronounced in tumors of animal not receiving SPG. The difference in the effectiveness of SPG immunotherapy when performed before and after PDT suggested that maximal interaction is achieved when immune activation peaks at the time of the light delivery or immediately thereafter. With SPG starting after PDT (and attaining an optimal immune activation 5-7 days later), it is evidently too late for a beneficial reaction.

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L7: Entry 10 of 47

File: PGPB

May 2, 2002

DOCUMENT-IDENTIFIER: US 20020052594 A1

TITLE: Method and kit for imaging and treating organs and tissues

Summary of Invention Paragraph (50):

[0047] Where normal organs or tissues are developed abnormally or are displaced in the body, or are insufficiently removed during ablative surgery, the tissue/organ-associated antibodies may be used as tissue-targeted vehicles for delivering therapeutic agents to said tissues in order to induce their involution or chemical and/or isotopic ablation. The antibodies or their fragments (or subfragments) can be conjugated with therapeutic modalities including, but not limited to, isotopes, drugs, toxins, photodynamic therapy agents, cytokines, hormones, autocrines, etc., which are used as cytotoxic or modulating agents, and which have hitherto been employed principally as toxic conjugates to cancer-targeting antibodies, as described in the reviews by Waldmann, T. A., Science 252:1657, 1991; Koppel, G. A., Bioconjug. Chem. 1:13, 1990; Oeltmann, T. N., and Frankel, A. E., FASEB J. 5:2334, 1991; and van den Bergh, H. E., Chemistry in Britain, May 1986, 430-439, incorporated herein in their entirety by reference.

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L7: Entry 31 of 47

File: USPT

May 1, 2001

DOCUMENT-IDENTIFIER: US 6224866 B1

TITLE: Immunotherapy of B cell involvement in progression of solid, nonlymphoid tumors

Detailed Description Text (6):

The immunotherapeutic composition may further comprise an additional component comprising one or more of a chemotherapeutic agent, an anti-inflammatory agent, an anti-B cell agent, or a combination thereof. Chemotherapeutic agents are well known in the art (e.g., see the description to follow for "drugs" which include exemplary chemotherapeutic agents). Anti-inflammatory agents, well known in the art, may be used to suppress the inflammation which contributes to tumor progression as described herein. Illustrative but non-limiting examples of anti-inflammatory agents include non-steroidal anti-inflammatory drugs (e.g., aspirin, ibuprofen, naproxen, and the like), and COX-2 inhibitors (e.g., rofecoxib, and celecoxib). The invention may be practiced with a preferred additional component to the exclusion of other additional components. The "anti-B cell agent" is an agent that acts directly against B cells, or acts indirectly (e.g., stimulates a Th1 response, thereby diverting away from a Th2 response may enhance B cell involvement in a pro-tumor immune response); wherein the anti-B cell agent comprises an immunomodulatory agent, or cytolytic agent, or a vector capable of encoding in a B cell an immunomodulatory agent or cytolytic agent that is subsequently introduced into B cells. In one embodiment, the additional component is administered separately (e.g., nonconjugated to; whether administered simultaneously, or administered separately, as part of a treatment regimen) with respect to the immunotherapeutic composition. In another embodiment, the additional component may be coupled to the affinity ligand of the immunotherapeutic composition; wherein the affinity ligand serves to selectively bind the B cells, thereby bringing the additional component in contact with or in functional proximity of B cells that may be involved in a pro-tumor immune response that promotes tumor progression. An immunomodulatory agent is an agent which, by interacting directly with such B cells or with naive CD4+ cells or with follicular dendritic cells, inhibits or prevents one or more of the following: activation and/or proliferation of B cells induced by the shed tumor antigen (e.g., shed tumor antigen by itself, or as presented to B cells by antigen presenting cells); secretion by plasma cells of anti-shed tumor antigen antibody which, in immune complex form, promotes tumor progression; a Th2 response. Such immunomodulatory agents may include, but are not limited to, a therapeutically effective amount of: cytokines, biologically active peptides, immunostimulatory sequences, drugs, and a combination thereof. The invention may be practiced with a preferred immunomodulatory agent to the exclusion of other immunomodulatory agents. For example, soluble CD21 has been shown to reduce antibody responses by blocking CD21 ligand on follicular dendritic cells and/or by binding C3 fragments associated with immune complexes (Qin et al., 1998, J. Immunol. 161:4549-54). Also, IL-12 has been shown to be a potent inducer of naive CD4+ cells towards a Th1 response (Palm et al., 1996/1997 Immunobiology 196:475-484; Jeannin et al., 1996, J. Immunol. 156:315903165). Further, certain short bacterial immunostimulatory DNA sequences ("ISS", containing unmethylated CpG motifs), have been shown to be able to stimulate a Th1 response (e.g., by inducing IL-12 production), and hence stimulate a cell-mediated immune response (Roman et al., 1997, Nat. Med. 3:849-854; Lipford et al., 1997, Eur. J. Immunol. 27:3420-3426). A cytolytic agent is an agent that, by interacting directly with such B cells, causes B cell cytotoxicity. Such cytolytic agents may include, but are not limited to, a therapeutically effective amount of: toxins; drugs; enzymes; cytokines; radionuclides; photodynamic agents; and molecules which induce apoptosis (e.g., Fas ligand). Toxins may include a therapeutically effective amount of ricin A chain, mutant Pseudomonas exotoxins, diphtheria toxoid, streptonigrin, boamycin, saporin, gelonin, and pokeweed antiviral protein. Drugs may include a therapeutically

effective amount of cytotoxic drugs including, but not limited to, fludarabine, chlorambucil, daunorubicin, doxorubicin (e.g., in liposomes), cisplatin, bleomycin, melphalan, mitomycin-C, and methotrexate. Due to the sensitivity of B cells to radiation, radionuclides may include, but are not limited to, radiometals such as yttrium which emits a high energy beta particle, and I.sup.125 that emits Auger electrons, that may be absorbed by adjacent B cells. Photodynamic agents may include therapeutically effective amounts of porphyrins and their derivatives. The methods for coupling ligands or targeting molecules with therapeutic agents are well known to those skilled in the art (See, for example, conjugates as reviewed by Ghetie et al., 1994, Pharmacol. Ther. 63:209-34; U.S. Pat. No. 5,789,554, the disclosure of which is herein incorporated by reference). Often such methods utilize one of several available hetero-bifunctional reagents used for coupling or linking molecules.

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File: USPT

May 1, 2001

US-PAT-NO: 6224866

DOCUMENT-IDENTIFIER: US 6224866 B1

TITLE: Immunotherapy of B cell involvement in progression of solid, nonlymphoid tumors

DATE-ISSUED: May 1, 2001

US-CL-CURRENT: 424/130.1; 424/134.1, 424/138.1, 424/141.1, 424/143.1, 424/152.1,  
424/153.1, 424/155.1

APPL-NO: 09/ 411116 [PALM]

DATE FILED: October 4, 1999

## PARENT-CASE:

This is a nonprovisional application based on earlier co-pending provisional applications Ser. Nos. 60/103,350 filed Oct. 7, 1998 and 60/117,526, filed Jan. 28, 1999 which are herein substantially incorporated by reference.

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